

Mesenteric Vascular Ischemia

Wilbert S. Aronow*

Cardiology Division, Department of Medicine, Westchester Medical Center/ New York Medical College, Valhalla, NY, USA

*Corresponding author: Wilbert S. Aronow, Cardiology Division, New York Medical College, Macy Pavilion, Valhalla, NY 10595, USA, Tel: (914) 493-5311; E-mail: wsaronow@aol.com

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Editorial

Elderly patients with underlying cardiovascular disease such as valvular heart disease, recent myocardial infarction, atrial fibrillation, and congestive heart failure are more likely to present with acute mesenteric vascular ischemia [1]. These disorders may lead to arterial macro embolization with sudden occlusion of mesenteric arteries [2]. Patients with chronic mesenteric vascular ischemia due to severe atherosclerotic obstruction of the mesenteric arteries may develop sudden thrombosis causing acute vascular ischemic symptoms [2].

Patients with acute mesenteric vascular ischemia usually present with severe steady abdominal pain out of proportion to the physical findings [2]. The pain is usually in the mid abdominal aorta. The patient may be writhing in pain unable to find a comfortable position. If peritoneal signs are present on physical examination, intestinal infarction has occurred. Mortality from mesenteric vascular ischemia is related to the state of the bowel at the time of diagnosis [3]. Bowel infarction greatly increases mortality and morbidity and has been associated with 80% mortality [3]. In a series of 49 patients with acute mesenteric vascular ischemia, the mortality rate was 65% [2].

Macro embolization from a proximal source such as atrial fibrillation or a myocardial infarction accounts for approximately one-third of causes of acute mesenteric vascular ischemia [3]. The superior mesenteric artery is the most common site of visceral arterial occlusion. Acute thrombosis of an already compromised vessel lumen due to preexisting atherosclerotic vascular disease accounts for another one-third of causes of acute mesenteric vascular ischemia [3]. More than 50% of patients who die from acute superior mesenteric artery thrombosis have a history of abdominal angina pectoris with postprandial abdominal pain and weight loss [3]. Abdominal angina pectoris typically occurs 15 to 60 minutes after eating and is associated with the amount of food eaten. In situ thrombosis typically occurs at the origin of the superior mesenteric artery and results in bowel infarction from the proximal jejunum to the mid transverse colon [3].

Nonocclusive mesenteric vascular ischemia occurs for most of the other causes of acute mesenteric vascular ischemia [3]. These patients typically have moderate- to-severe mesenteric atherosclerotic disease with a marginal cardiac reserve. Administration of vasoactive drugs such as digoxin further exacerbates the situation causing acute hemorrhage and bowel necrosis [4]. An acute reduction in cardiac function may reduce mesenteric perfusion pressure. Paradoxical splanchnic vasoconstriction may follow with micro vascular collapse, formation of micro thrombi, and capillary sludging causing acute mesenteric vascular ischemia.

An uncommon cause of acute mesenteric vascular ischemia is mesenteric venous thrombosis [5]. These patients may have a history of associated deep venous thrombosis of the lower extremities or hypercoagulable disorders, many nondiagnostic laboratory

abnormalities occur in patients with acute mesenteric vascular ischemia and infarction. These abnormalities include hemoconcentration, leukocytosis with a left shift, metabolic acidosis, hyperphosphatemia, and hyperamylasemia [2].

Abdominal roentgenograms may exclude mechanical small bowel obstruction or perforation of a hollow viscus as the cause of abdominal pain. The majority of patients with acute mesenteric vascular ischemia will show one of the following signs on abdominal roentgenograms: ileus, ascites, small bowel dilation, thickening of valvulae conniventes, and separation of small bowel loops [2]. Occasionally, a gas-less abdomen may be seen due to excessive fluid accumulation within the lumen [2].

Arteriography requires hemodynamic stabilization of the patient since hypotension can cause splanchnic vasoconstriction and preclude an adequate study. Vasoactive drugs with splanchnic vasoconstrictive properties cannot be used. The anterior/posterior (AP) view best shows collateral vessels [2]. Lateral aortography best visualizes the origins of major visceral arteries that overlie the aorta in the AP plane [2].

Generally, arteriographic signs can differentiate embolic from thrombotic occlusions [6]. Emboli to the superior mesenteric artery usually are just proximal or distal to the origin of the middle colic artery and may be associated with minimal atherosclerotic lesions. Thrombotic occlusions of preexistent stenotic lesions occur more commonly at the origin of the superior mesenteric artery and are associated with generalized atherosclerosis of the aorta and with extensive collaterals. Mesenteric venous thrombosis shows slowing of arterial blood flow and nonopacification of the corresponding mesenteric or portal veins. Nonocclusive mesenteric vascular ischemia shows narrowing and irregularity of major branches of the superior mesenteric artery [2].

Nonocclusive mesenteric vascular ischemia may be treated with intra-arterial infusion of a vasodilator into the superior mesenteric artery [6,7]. Surgery may be avoided in these patients if the diagnosis is clear on arteriography and abdominal symptoms and signs completely resolve with infusion of papaverine.

All patients with suspected embolic or thrombotic occlusions causing acute mesenteric vascular ischemia must undergo exploratory celiotomy. Intravenous fluids, infusion of continuous anticoagulation with heparin, and broad spectrum antibiotics are indicated prior to surgery [2]. Usually an embolus can be directly extracted from the superior mesenteric artery. If thrombosis of an atherosclerotic lesion is suspected, or if extraction of the embolic material is not possible or incomplete, an aortomesenteric bypass should be considered. The operative approach to venous thrombectomy involves resection of the infarcted bowel [2] of 40 patients with acute mesenteric vascular ischemia, 38 (95%) were treated with surgery and 2 (5%) with thrombolysis in the superior mesenteric artery [8]. Of the 40 patients,

20 (50%) died. However, the 15 patients treated within 16 hours after the onset of symptoms all survived [8].

The basic treatment of acute mesenteric vascular ischemia is early recognition, resuscitation, urgent revascularization, resection of necrotic bowel, and reassessment with second-look laparotomies [9]. Endovascular techniques now offer a less invasive alternative, but whether an endovascular-first or open surgery-first approach is preferred for most patients is unclear [9].

Chronic mesenteric vascular ischemia is associated with atherosclerotic occlusions or stenoses [10]. Because of abundant collateral vessels, multiple vessel involvement is usually needed before classic postprandial abdominal pain and weight loss occur. Patients frequently complain of bloating and early satiety. Weight loss may be severe if the patient develops severe incapacitating abdominal pain after eating. Chronic mesenteric vascular ischemia may present as exercise-induced abdominal pain [11].

The diagnosis of chronic mesenteric vascular ischemia is based on symptoms caused by visceral occlusive arterial disease, exclusion of other gastrointestinal causes of the symptoms, and arteriographic demonstration of occlusive lesions and development of collaterals. Vascular reconstruction can be accomplished by Enderterectomy or aortomesenteric bypasses [12] of 54 patients with chronic mesenteric vascular ischemia, 43 patients were treated with endovascular revascularization and 11 patients with open surgical revascularization [13]. The symptoms were abdominal pain in 98% of the patients, weight loss in 53% of the patients, and diarrhea in 25% of the patients. Computed tomography angiography was the key diagnostic tool for 60% of the patients. Endovascular and open surgical revascularization had similar early and late outcomes [13].

Eighty-six patients with chronic mesenteric vascular ischemia had open surgical treatment [14]. Median follow-up was 6.9 years (range 0.3-20 years). The 3-day mortality was 3.5% and the 30-day morbidity 13.9%. Ten-year survival was 88% for patients with complete revascularization and 76% for patients with incomplete revascularization. Freedom from digestive symptoms at 10 years was 79% for patients with complete revascularization and 65% for patients with incomplete revascularization [14].

A meta-analysis was made of 12 studies comparing endovascular revascularization versus surgical revascularization for the management of chronic mesenteric vascular ischemia [15]. The primary endpoint of perioperative (30 days) survival was not significantly different between both revascularization procedures. A secondary endpoint of perioperative mortality, nonfatal cardiac events, nonfatal stroke, and nonfatal bowel ischemia was not significantly different between both

revascularization procedures. The cumulative odds ratio for survival after the thirtieth day was not significantly different between both revascularization procedures. Late primary patency was reported in 8 of the 12 studies with a cumulative odds ratio of 3.57 (95% CI, 1.83-6.97; $p = 0.0002$) favoring surgical revascularization [15].

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